

Later, when systemic chemotherapy was considered, we again had to proceed cautiously. We gave prophylactic gamma globulin just before chemotherapy in an attempt to prevent infectious complications (this patient was already immunosuppressed due to asplenia and CLL). His hematocrit was extremely low at 15 to 17%.

He responded moderately to erythropoietin (prechemotherapy hematocrit ~20%). Even so, he required a transfusion of packed red blood cells when his hematocrit rapidly dropped to 11% shortly after the chemotherapy. Ideally, we would have liked to prevent such a extreme drop in hematocrit. Frequent monitoring of blood count and clinical status cannot be overemphasized. The response of the CLL was excellent, however, and with aggressive monitoring and supportive care, the patient tolerated the therapy.

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## Suppurative Parotitis

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SUPPURATIVE PAROTITIS, an uncommon condition of the elderly and severely ill, is often associated with dehydration and sialolithiasis.<sup>1,2</sup> We report an unusual case featuring a mixed infection of *Candida albicans* and *Streptococcus pyogenes* and review the history and management of suppurative parotitis.

## Report of a Case

A 19-year-old man had a 3-week history of polyuria and polydipsia and a 3-day history of fever and right-sided facial swelling and pain. His medical history was unremarkable except for a left parotid duct stone without parotitis, which was treated conservatively when he was 5 years old. Upon physical examination, he was dehydrated, his breath had a fruity odor, and his right parotid gland was swollen, tender, and erythematous. No discharge from Stensen's duct was noted. Facial nerve function was normal bilaterally throughout the clinical course. Laboratory results showed a total white blood cell count of  $17 \times 10^9/L$ , blood glucose 1529 mg/dL, positive serum ketones, and arterial pH 7.11; HIV serology was negative.

The patient was given intravenous fluids and insulin, and therapy was begun with oral amoxicillin/clavulanate for presumed parotitis. After 48 hours, his ketoacidosis and hyperglycemia had resolved, but he still had a fever and a swollen parotid gland. Amoxicillin/clavulanate was discontinued and intravenous nafcillin therapy begun. The next day, a computed tomography (CT) scan revealed intraparotid fluid collection. Purulent material (10 cc) was drained through an operative incision; staining of the fluid revealed few gram-positive cocci and moderate budding yeast in chains, and the cultures subsequently grew both *Candida albicans* (heavy) and *Streptococcus pyogenes* (moderate). Antibiotic therapy was changed to intravenous cefuroxime.

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After 72 hours of cefuroxime, the patient remained febrile with persistent swelling and erythema of the parotid incision. A second CT scan failed to demonstrate any fluid collection and, in view of the culture results, therapy with intravenous amphotericin B was begun. Within 48 hours, the patient's fever, swelling, and erythema resolved.

### Etiology and Presentation

Acute suppurative parotitis affects predominantly the elderly and severely ill. In a 1962 study,<sup>2</sup> 131 of 161 patients developed parotitis in the context of severe or multiple diseases. Most patients were more than 60 years of age; 43% were older than 70. Common predisposing features were preexisting oral disease (77%), dehydration (50%), malnutrition (68%), and the use of medications that reduce oral secretions (25%).<sup>2</sup>

Given the correlation of dehydration and antisialogogues with parotitis, one mechanism of parotitis may be retrograde transductal migration of oral microorganisms in the context of either decreased salivary flow or partial obstruction by a sialolith.<sup>2</sup> Underlying oral disease predisposes to changes in mucosal flora, as does bacterial overgrowth of the oral cavity, which has been documented in states of impaired salivation. The altered oral flora contribute to infection as mucosal surfaces are colonized by microorganisms with enhanced pathogenicity. This model—change in oral flora with subsequent ascending infection—fits well with the isolation of *Candida* species in our patient. It may be that oropharyngeal candidiasis associated with uncontrolled diabetes<sup>3</sup> and decreased salivary flow associated with the dehydration of ketoacidosis provide the factors necessary for the development of parotitis.

*Staphylococcus aureus* is the most common pathogen isolated in suppurative parotitis; alpha hemolytic streptococci and enteric gram-negative rods have been documented in a minority of cases. In 1961, Krippaehne et al. noted that of 66 parotid glands, 41 contained staphylococci alone; 20 contained both streptococci and staphylococci; eight contained gram-negative bacteria; and two contained pneumococci.<sup>2</sup> In contrast, a recent study using modern anaerobic culture techniques<sup>4</sup> found that of 36 isolates from patients with suppurative parotitis, 20 contained anaerobic bacteria and only 16 contained the traditional pathogens of staphylococci, streptococci, or gram-negative bacilli. The anaerobes isolated were predominantly oral anaerobic organisms such as *Peptostreptococcus* sp. and *Fusobacterium* sp.;<sup>4</sup> unusual cases of parotitis with *Treponema pallidum*, *Mycobacterium* sp., *Eikenella* sp., and *Bartonella* sp. have been documented.<sup>1</sup> Fungal parotitis in the context of diabetes has also been reported in an elderly woman who devel-

oped a parotid abscess with *Torulopsis glabrata*.<sup>5</sup>

Suppurative parotitis presents relatively acutely with the onset of unilateral swelling, erythema, and tenderness of the parotid gland. Fever, leukocytosis, and other signs of systemic illness are common. Intraorally, the parotid duct is often visible and edematous, and it may have a purulent discharge at the orifice. Abscess formation is common and clinically difficult to distinguish from simple parotitis, as the dense parotid capsule prevents extension of purulence to the skin. Complications, both systemic and local, are common. Bacteremia and subsequent systemic sepsis with multisystem organ failure and death have been reported.<sup>2</sup> Locally advanced disease can penetrate anteriorly to the skin and soft tissues of the face, inferiorly to the planes of deep tissue in the neck where abscess formation and septic thrombophlebitis of the jugular vein can occur, or posteriorly to the mandible, temporomandibular joint, or middle ear and external auditory canal. Facial nerve palsy may result, both as a consequence of the local infection and as a complication of surgical interventions.

### Management

Medical therapy alone is usually successful in suppurative parotitis; surgery is reserved to manage local complications and failures of medical management. The series of Krippaehne<sup>2</sup> recorded the need for surgical intervention in 47 of 161 cases, usually after a trial of medical therapy. Before antibiotic therapy is begun, the clinician should obtain adequate culture material, including any drainage from Stensen's duct, to guide organism-specific therapy. Fine-needle or surgical aspiration may be required to obtain an adequate sample, which is cultured for aerobic bacteria, anaerobic bacteria, and fungus infection. Initial therapy is directed at staphylococci and streptococci and then modified according to the culture results and response to therapy.<sup>1</sup> Suitable regimens include intravenous nafcillin and oral dicloxacillin; clindamycin is an alternative for the patient allergic to penicillin. In the event of suboptimal clinical response to therapy, imaging studies such as CT or sonography may identify the presence of a parotid abscess, which mandates early and aggressive drainage. In the absence of such a focal collection, a more exhaustive microbiological search is appropriate.

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